Challenging Plaintiff's Expert

Defending Lead-Containing Toy Lawsuits

By Ryan L. Nilsen and Michael R. Carey

Think creatively and exploit as many weaknesses as possible.

An injured child epitomizes the sympathetic plaintiff. Not surprisingly, therefore, children injured by toys containing lead have captured the attention of the plaintiffs' bar. Proving—or disproving—exposure, causation, and damages in lead poisoning cases, however, is not child's play.

This article will explore ways to challenge plaintiffs' experts in lead toy exposure litigation by highlighting common weaknesses in exposure analysis, cognitive-injury proof, and specific causation.

Lead Is Everywhere

Lead is everywhere in our environment. Its most prevalent source is leaded-fuel exhaust that, for decades, has accumulated in the soil and entered our bodies as dust. Exposure also occurs in residential homes when lead paint peels and flakes off walls and children ingest the chips or when drinking water becomes contaminated by lead piping and solder used in home plumbing.

Because lead is so prevalent in the environment, nearly everyone has a measurable blood-lead level (BLL). According to the Centers for Disease Control and Prevention (CDC), the mean level of lead in children ages one to five was 2.2 µg/dl in 2000. Any level higher than 10 µg/dL, the CDC notes, is cause for concern.

Lead has no known biological role in the body. Its toxicity results from its ability to mimic other biologically important metals, such as calcium, iron, and zinc, that bind to proteins and molecules throughout the body. Once bound to lead, these proteins and molecules can no longer carry out the same reactions, such as producing enzymes necessary to certain biological processes.

For centuries, lead exposure has been associated with negative health effects, including irreversible neurological damage, renal disease, cardiovascular effects, and reproductive toxicity. According to modern scientists, famed composer Ludwig van Beethoven died of lead poisoning.

Among United States toy manufacturers, lead content is regulated by the Consumer Product Safety Commission (CPSC). CPSC

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regulations apply to everything from metal alloys used in toy jewelry to the paint that covers the toys themselves. But CPSC regulations do not apply to the manufacture of toys abroad. Nevertheless, domestic distributors and retailers remain liable under state product liability laws for the harmful effects of foreign-made products that they place into the stream of commerce.

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Evidence of Exposures Is Not Proof of Causation

Various factors affect proof of specific causation in lead-exposure cases, including exposure, absorption, pathway, and absorption rates. Look for four things to attack plaintiff's proof of specific causation. The first element of specific causation is actual exposure to lead. In lead, as in other toxin cases, exposure is a condition precedent to causation. Exposure can be proven in a variety of ways. For example, blood tests can measure suspected lead exposure. When elevated levels of lead in the bloodstream are present, the plaintiff's treating physician can testify to the lead levels found in the plaintiff's blood and the symptoms reported.

But proof of exposure is not enough to establish specific causation. Proof that a toy caused lead poisoning also requires evidence that the lead present in the toy is in a form that can be absorbed into the body. For this type of evidence, a plaintiff must offer expert testimony, often from a toxicologist. Be skeptical of a treating doctor, or a professed exposure expert, who merely testifies that the toy at issue contains lead and that children generally put toys in their mouths. That children put toys in their mouths does not prove that a toy containing lead can or actually did transfer lead into a child's body or in an amount consistent with the level of lead found in the child's blood. Lead absorption is a function of lead's physiochemical properties, concentration, and commingling with other agents that can assist or interfere with its intake. "For example, inhaled lead is absorbed almost totally, whereas ingested lead is taken up only partially into the body. Iron deficiency and low nutritional calcium intake, both common conditions of inner-city children, increase the amount of ingested lead that is absorbed in the gastrointestinal tract and passes into the bloodstream." B. Goldstein and M. Henifin, *Reference Guide on Toxicology*, Reference Manual on Scientific Evidence (2d ed. 2000) at 424.

In addition to exposure and absorption, an expert must be prepared to explain the pathway of exposure from the toy into the plaintiff's body. The primary pathway for lead poisoning is ingestion. But lead can also enter the body through skin absorption and, less frequently, inhalation. An expert must account for the method of delivery—and based on scientific principles, not mere speculation. In this regard, watch out for the exposure expert who concludes that ingestion occurred and then sets out to find support for the conclusion. *See Mitchell v. Gencorp Inc.*, 165 F.3d 778, 783 (10th Cir. 1999) (recognizing that scientists who testify about their conclusions before performing necessary validation tests may lack the objectivity required by the scientific method).

Proof of the pathway of exposure will, in turn, require evidence excluding other sources of lead exposure. Adults most often encounter lead at work, while children generally encounter lead through play. Children generally absorb lead into their bloodstream at a higher rate than adults. For both adults and children, the main sources of lead poisoning are lead-contaminated soil and lead-based paint dust or chips, particularly in older houses. Lead-based paints can disintegrate into powder and be inhaled or swallowed, such as when teething children suck on painted windowsills as they look outside. Lead dust can also enter the circulatory system through the digestive track faster and more readily than a solid piece of lead.

Finally, proof of specific causation requires evidence on lead absorption rates. Almost 100 percent of inhaled lead is absorbed into the body, compared to just 20 to 70 percent of ingested lead (with children generally absorbing higher percentages than adults). Accordingly, the absorption of lead from a lead painted toy is different from a toy made of lead.

Regulatory Standards Are Not Injury-Causation Thresholds

The key to understanding the injury-causation thresholds is the relationship between exposure level and injury risk. As a basic tenet of toxicology, the injury resulting from a toxin depends on the dose of the toxin received. Related to that tenet is the concept of "threshold dose." The "threshold" or "no effect level" is the level of exposure below which a substance does not exhibit a toxic effect or at least manifest a clinically observable effect.

In 1975, blood-lead levels up to 30 µg/dl were considered safe. In 1985, the threshold was lowered to 25 µg/dl. In 1991, the threshold was lowered again to the present standard of 10 µg/dl.

In response to recent concerns over lead in imported toys, the CDC has shrugged off calls for further reduction of its lead threshold level because, according to the CDC, "any decision to establish a new level of concern would be arbitrary and provide uncertain benefits."

Thus, regulatory standards do not provide compelling evidence of injury-causation thresholds for litigation. Regulatory standards "traditionally include protective factors to reasonably ensure that susceptible individuals are not put at risk." B. Goldstein and M. Henifin, *Reference Guide on Toxicology*, Reference Manual on Scientific Evidence (2d Ed. 2000) at 424. The mere fact that an individual has been exposed to a level above a regulatory standard does not necessarily mean that an adverse effect has occurred.

Nevertheless, plaintiff experts use regulatory standards to prop up their causation theories. When a child's BLL exceeds 10 µg/dl, they argue, adverse health effects will follow. Indeed, research conducted since 1991 offers some evidence that children's physical and mental development can be affected at BLLs below 10 µg/dL.

But the CDC and EPA's choice of 10 µg/dL is based on a significant body of scientific evidence showing that several significant health

effects occur in the 10–15 µg/dL range. The EPA emphasizes, however, that its standard does not imply that 10 µg/dL is a threshold level. On the contrary, EPA maintains that there is no known threshold for lead, and that it chose 10 mg/dL because studies of adverse health effects at lower levels of exposure are not well substantiated and observed only a limited number of children.

Plaintiffs claim that adverse health effects arise at blood-lead levels far below the 10 µg/dL regulatory standard, and their experts rely on the hazard standard to suggest very low-dose exposures cause injuries in children. Reliance on the hazard standard is especially prevalent in lead toy cases in which the uptake of lead is difficult to quantify.

If confronted by this claim, defendants should emphasize that neither the CDC nor the EPA has lowered its hazard standard. Unless and until the CDC or the EPA changes their hazard standard, the defense bar should attack any expert who opines on specific causation based on exposures below the 10 dl/mg threshold. For example, a New York court recently relied upon the 10 µg/dl hazard standard to find no negligence in a medical malpractice case for failing to treat a patient with a BLL of 9 µg/dL. *See Breeden v. Valentino*, 2007 WL 3070774 *2 (N.Y. Sup. 2007). The court noted that, under New York State Public Health Law, lead poisoning is not diagnosed in children until the blood lead level reaches 10 µg/dL.

Courts Insist on Proof of Causation

In lead exposure litigation, proof of specific causation is the plaintiff's single biggest stumbling block to recovery. The New Jersey Supreme Court described it this way:

> By far the most difficult problem for plaintiffs to overcome in toxic tort litigation is the burden of proving causation. In the typical tort case, the plaintiff must prove tortious conduct, injury and proximate cause. Ordinarily, proof of causation requires the establishment of a sufficient nexus between the defendant's conduct and the plaintiff's injury. In toxic tort cases, the task of proving causation is invariably made more complex because of the long latency period of illnesses caused by carcinogens or other toxic chemicals. The fact that ten or twenty years or more may intervene between the exposure and the manifestation of disease highlights the practical difficulties encountered in the effort to prove causation.

James v. Bessemer Processing Co., 714 A.2d 898, 909 (N.J. 1998).

Poking holes in specific causation is the defendant's best chance to defeat a lead exposure lawsuit. Specific causation is the direct connection between two events in which the occurrence of one event is necessary to the event that follows.

Epidemiology alone cannot prove specific causation. It can only demonstrate that lead is *capable* of causing certain harms. For this reason, defense counsel should be on guard against any expert who plans to opine on specific causation based solely on the fact that lead exposure can cause harm. Also untrustworthy are causation opinions connecting high BLLs to nothing more than a child's toy-box full of lead-containing toys, without addressing alternative explanation. With so many pathways for exposure, the expert who concludes that a lead-containing toy is the source of elevated BLLs is ripe for attack. From the beginning of a case, manufacturer defendants should prepare for a full-scale *Daubert* attack under Federal Rule of Civil Procedure 702.

Courts require proof of exposure with varying degrees of specificity. But one thing is common in all courts: some effort to quantify the exposure is required. In *Cartwright v. Home Depot U.S.A., Inc.*, 936 F. Supp. 900, 902 (M.D. Fla. 1996), the plaintiff's expert toxicologist opined that the defendant's paints caused his asthma. While the plaintiff's toxicologist identified several components of the paints that were known respiratory irritants, he provided no information as to how much of the particular components the plaintiff had been exposed. Likewise, he failed to "provide any quantification to substantiate in scientific terms what level of exposure would have been sufficient to cause asthma in the plaintiff or anyone else." *Id.* at 904. The court granted the defendant's motion to exclude the plaintiff's toxicology evidence, stating:

> Plaintiffs cite no authority for the propositions that irritating chemicals in latex paints become bioavailable in relevant amounts, that actual exposure levels from any particular uses of latex paint are high enough to cause any reaction, that prolonged, unspecified low level exposure to irritants can cause asthma, or that latex paints generally (or these paints in particular) cause asthma.

Id. at 905.

Be Wary of the Overreaching Neuropsychologist

Some lead exposure studies have reported cognitive declines from quantifiable exposures. Other researchers and child advocacy groups claim an association between early lead exposure and extreme learning disabilities, including speech development and even autism. Some studies even attribute behavioral problems to lead exposure. Plaintiffs frequently offer the testimony of a neuropsychologist to prove causation.

Predictably, therefore, plaintiffs may rely on a neuropsychologist to establish specific causation between lead ingestion and neuro-cognitive deficiency. More sophisticated plaintiffs may proffer a neuropsychologist with a specialty in pediatrics.

Defendants should be skeptical of a neuropsychologist who attempts to link cognitive deficits to lead exposure based on the alleged onset of reduced IQ. Rarely will a neuropsychologist have conducted an IQ test before the alleged exposure to use as a baseline. And without a baseline test, the expert's causation opinions are susceptible to exclusion.

In *Palmer v. Asarco Inc.*, 510 F. Supp. 2d 519, 530 (N.D. Okla. 2007), the defendants challenged a neuropsychologist's causation opinion on the ground that she "must know what plaintiffs' educational abilities would be without any exposure to lead in order to testify." The court agreed that "[t]his is true for certain types of injuries, such as IQ loss because, without pre-exposure testing, any opinion that plaintiffs have lost IQ points is purely speculative." *Id.* Thus, while the expert could testify that the plaintiff has a cognitive deficit as compared to the general population, she could not testify that the deficit is attributable to lead exposure.

Similarly, in *Adams v. Rizzo*, 831 N.Y.S.2d 351, 2006 WL 3298303 (N.Y. Sup. 2006), the court highlighted a common mistake of plaintiffs in attempting to prove specific causation:

Judging from these statements and comments made by plaintiffs' counsel during depositions and at oral argument, it appears that plaintiffs hold the view that once constructive or actual notice is established together with "elevated blood lead levels" any and all injuries, disabilities, syndromes or cognitive-intellectual-behavioral problems are exclusively and dispositively linked to lead exposure and the defense is foreclosed from discovery on the issue of damages and neurodevelopmental problem causation. This court disagrees.

Regulatory standards do not provide compelling evidence of injury-causation thresholds for litigation.

Id. *12. The court instead concluded that "[t]he generally accepted view that lead is a health hazard and may have an association with the alleged injuries does not equate with prima facie proof that lead exposure was a substantial factor in causing the injuries alleged herein to *these* plaintiffs." *Id.* (emphasis added).

The expert must explain why exposure to a lead-containing toy—and not some other environmental factor—caused the plaintiff's elevated blood-lead levels. Failure to weigh alternative causes for a plaintiff's injury is proper grounds for excluding a plaintiff's specific causation testimony. In *Bunch v. Artz*, 2006 WL 2411428 *1 (Va. Cir. Ct. 2006), a lead paint case, the defendants sought discovery requesting that the "mother, Ms. Bunch, provide '[e]ducational history of parents and siblings, and potentially other family members if there is a history of learning problems or special education.'" *Id.* (citations omitted). The court granted the request, finding that the evidence was relevant to "determining whether any deficits the plaintiff may have are solely the result of lead poisoning or whether other factors, such as his mother's intelligence and education, are contributing factors." *Id.* Similarly, the Second Circuit Court of Appeals upheld the rejection of proffered expert testimony in *Wills v. Amerada Hess Corp.*, 379 F.3d 32 (2d Cir. 2004), because the expert failed to weigh alternative explanations. In that case, the widow and executrix for a seaman who died of cancer alleged that her husband's cancer had been caused by exposure to benzene and other alleged carcinogens during his work aboard the defendants' vessels. The expert failed to account for other cancer risks in his conclusions—in particular, cigarette smoking and alcohol consumption—and the court struck his opinions. *Id.* at 50.

Defense practitioners should be on the lookout for novel theories unsupported by reliable scientific literature. The court in *Palmer v. Asarco Inc.* excluded the causation testimony of a neuropsychologist as unreliable. The proposed testimony alleged a link between lead exposure and ADHD. *Id.* at 531. However, the court described this testimony as "a novel scientific theory and there does not appear to be any support in the scientific literature for [the doctor's] claim that lead exposure is a recognized cause of ADHD." *Id.* According to the court, even if the doctor "believes this link exists, without scientific support and research cited in her expert report and deposition this opinion is classic *ipse dixit*, and any testimony that lead exposure caused ADHD will be excluded." *Id.* Similarly, in *Veloz v. Refika Realty Co.*, 831 N.Y.S.2d 399, 2007 N.Y. Slip Op. 01980 (N.Y. App. Div. 2007), an expert failed to support his general causation opinion because he "[did] not cite any particular scientific literature, nor [did] he identify which impairments were so described, who so described them, the similarity of those so described to those he saw in plaintiff, and at what level of exposure to lead such impairments have been observed."

Rein In the Treating Physician

Like the overreaching neuropsychologist, courts often are skeptical of treating physicians who opine about causation in toxic exposure cases. Defense practitioners should closely scrutinize the opinions of treating physicians, looking for causation opinions.

Most treating physicians fail to consider the dose-response relationship, exposing themselves to a *Daubert* challenge. In *Farris v. Intel Corp.*, 493 F. Supp. 2d 1174 (D.N.M. 2007), the defendants challenged the causation opinions of the plaintiff's general practitioner. The court noted that clinical experience does not provide support for an opinion requiring experience in epidemiology or toxicology. *Id.* (citing *Siharath v. Sandoz Pharms. Corp.*, 131 F. Supp. 2d 1347, 1351 (N.D. Ga. 2001)).

In framing the *Daubert* attack on treating physicians, defense counsel should focus on the treating doctor's lack of qualifications in the areas of toxicology and epidemiology. Despite otherwise impressive résumés, clinical practitioners generally are unqualified to opine about causation issues in lead exposure cases, especially when the treating physician has not published articles or case studies or conducted any other research on the effects of lead exposure or has limited experience in treating patients with lead exposure injuries. *See Leathers v. Pfizer, Inc.*, 233 F.R.D. 687 (N.D. Ga. 2006).

In the majority of federal circuits, experts must be qualified on an opinion-by-opinion basis. More and more courts disallow treating physicians to use their medical degrees to opine beyond their expertise. Having a medical degree is not sufficient to qualify an expert to testify about every medical question. *O'Conner v. Commonwealth of Edison Co.*, 807 F. Supp. 2d 1376, 1390, *aff'd*, 13 F.3d 1090 (7th Cir. 1994); *see also Porter v. Whiehal Lab., Inc.*, 9 F.3d 607, 614–15 (7th Cir. 1993) (excluding a doctor's causation opinion because the doctor admitted that the area in question was outside his expertise). Courts require experts to have "special knowledge" to opine on a topic within a sub-specialty of a discipline.

Limit the Toxicologist

Specific causation generally falls within the expertise of a toxicologist because toxicology is "the study of adverse effects of chemicals on living organisms." Casarett and Doull's Toxicology: The Basic Science of Poisons 13 (Curtis D. Klassen ed., 5th ed. 1996).

But even toxicologists may be limited in their ability to opine about the level of lead absorption into the bloodstream from different types of lead-containing toys. One of the inherent characteristics

of lead is that it leaves no identifiable tracers; thus, identifying the specific source of lead found in a plaintiff's bloodstream is usually impossible. According to one expert, the "bioavailability [of lead] operates via a set of biological, biochemical and physico-chemical processes that will often render starting forms of lead in pigments indistinguishable in toxicokinetic terms." *Thomas v. Mallett*, 701 N.W.2d 523, 536 (Wis. 2005). Because the uptake, distribution, retention, and excretion of lead is identical whatever the lead's source, a toxicologist who claims to be able to identify the source of lead responsible for the harm based solely on blood contamination is subject to exclusion.

Just like the treating physician, a toxicologist may have an impressive résumé, one that suggests sufficient training, skill and experience to practice toxicology, but still be disqualified in your lead exposure case if he or she lacks specific knowledge and experience about lead exposure. *See*, *e.g.*, *Wintz v. Northrop Corp.*, 110 F.3d 508, 514 (7th Cir. 1997) (finding a toxicologist unqualified to testify regarding an infant's abnormalities because the expert was not a licensed physician, lacked expertise in genetic disorders, and was inexperienced with the toxin at issue). Thus, if the plaintiff's toxicologist lacks experience in lead poisoning, particularly in children, defendants should challenge the expert's qualifications.

Finally, under *Daubert*, a toxicologist must employ the same level of scientific rigor in the courtroom as he or she would in the laboratory. In a lead toy case, a toxicologist should at least rely on materials and studies based on lead poisoning among *children*. While there are numerous materials arising out of lead-based paint litigation used in homes, concern over lead in toys is a relatively recent phenomenon. As a result, "scientific" studies in this area are scarce. Thus, a toxicologist in a lead toy case who analogizes his or her opinions to lead paint exposure may reach too far. Ingestion of lead-paint chips is quite different from a child ingesting lead from toys.

Conclusion

Recent product liability cases suggest some defenses to expert testimony proffered in lead-exposure litigation. The practitioner should:

- Consider whether the expert's testimony relates to scientific, technical, or other specialized knowledge;
- Determine whether the proffered testimony will assist the trier of fact;
- Determine whether the expert is qualified;
- Use the traditional *Daubert* factors as a starting point and decide whether the factors are appropriate;
- Consider whether other factors, not mentioned in *Daubert*, are relevant;
- Consider whether the expert's opinion is:
  - based upon facts consistent with the undisputed facts;
  - supported by reliable source data and supportable, reasonable assumptions of the type normally relied upon by experts in the field;
  - based upon an approach that employs the same level of intellectual rigor that characterizes the practice of the expert in the relevant field.

Exclusion of expert testimony can be achieved by finding and focusing on a weakness in the expert's person and/or opinion. It is important for practitioners to think creatively and to exploit as many of those areas as possible. In the context of lead exposure, due to its indeterminate nature, savvy defendants should have a lot of arrows in their quivers. FD